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Lung crackle characteristics in patients with asbestosis, asbestos-related pleural disease and left ventricular failure using a time-expanded waveform analysis – a comparative study

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The aim of this study is to investigate lung crackle characteristics by time-expanded waveform (TEW) analysis in patients with asbestosis (AS), asbestos-related pleural disease (ARPD) and left ventricular failure (LVF). TEW was performed on a 33 s recording from each of 40 patients (12 AS, 17 ARPD and 11 LVF). They were 38 men and two women. Crackles on TEW were counted during inspiration and expiration, and the timing of clusters of crackles with respect to inspiration and expiration was noted. A total of 1117 crackles were identified. The initial deflection width (IDW) and the two cycle duration (2CD) were calculated for all crackles within one respiratory cycle for each patient (total of 298 crackles).

Crackles were detected by TEW in all patients with AS, in seven patients with ARPD and in nine patients with LVF. Crackles in AS were mainly fine, mid- to late-inspiratory. Crackles in LVF took three patterns; in the first there were repetitive mid- to late inspiratory crackles similar to those seen in AS except that the crackles in LVF tended to be medium and coarse as well as fine (three patients); in the second crackles started early in inspiration followed by a crackle-free period then by another cluster of crackles lasting to the end of inspiration and to the early third of expiration (four patients) and in the third there were repetitive expiratory crackles with no or few inspiratory crackles (two patients). Crackles in ARPD generally took the configuration of fine crackles but another type of crackle preceded by a sharp deflection followed by an M-shape oscillation then by the largest oscillation was also found.

IDW and 2CD for inspiratory crackles in ARPD were shorter than those in AS and LVF (for IDW $P < 0{\cdot}009$ and $P < 0{\cdot}003$ compared with AS and LVF respectively and for 2CD, $P < 0{\cdot}006$ and $P < 0{\cdot}003$ compared with AS and LVF respectively). IDW and 2CD in AS tended to be shorter than these for LVF but these results did not reach statistical significance. It is concluded that many differences exist between crackles in AS, LVF and ARPD. Differences in nature and timing of crackles may reflect differences in the pathophysiology and mechanism giving rise to lung crackles in these conditions. TEW provides informations of diagnostic value.

Introduction

Crackles over the lung bases are commonly the only clinical signs of asbestosis. Crackles in asbestosis (AS) are described as mid- to late- inspiratory, fine, high pitched and localized on the lower and middle lung zones (1,2). Asbestos workers have usually been smokers and often have ischaemic heart disease and/or hypertension which may lead to left ventricular failure (LVF). Lung crackles in early left ventricular failure are clinically difficult to distinguish from those heard in asbestosis and differentiation between asbestosis and left ventricular failure may be a clinical problem.

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In patients with acute pleurisy an inspiratory and expiratory adventitious sound called a 'friction rub' can be heard and also detected by a time-expanded waveform (TEW) analysis (3). This sound is thought to be due to the contact between the roughened surfaces of the two layers of pleura during inspiration and expiration. In patients with asbestos-related pleural disease (ARPD) adventitious mid-inspiratory and mid-expiratory 'clicking' sounds are often audible (our observation), but the TEW features of this sound have not been documented. These sounds may give rise to a clinical suspicion of asbestosis of the lung parenchyma.

In this study, lung crackle characteristics including their distribution, timing during the respiratory cycle, their initial deflection width (IDW) and two cycle duration (2CD) are investigated in patients with AS, LVF and ARPD.

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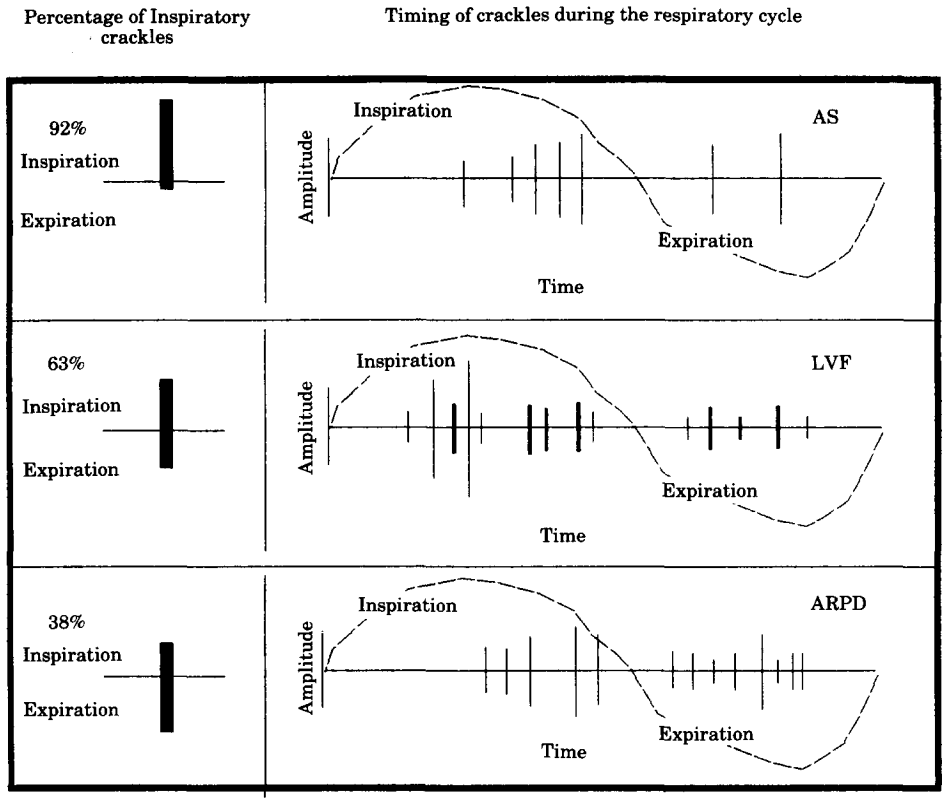

Fig. 1 Summary of the distribution of crackles for AS, LVF and ARPD. On the left, percentage of crackles in inspiration and expiration. On the right, distribution of crackles during the respiratory cycles (see also Table 2). The thin and thick lines symbolize fine and coarse crackles respectively.

Patients

Forty patients were studied (12 with AS, 11 with LVF and 17 with ARPD). All patients with AS had substantial asbestos exposure and the diagnosis of asbestosis was based on the presence of interstitial opacities consistent with asbestosis on high resolution computed tomography (HRCT). All patients had pleural disease of less than 2b grade on the ILO score (length of pleural disease is less than one quarter of the chest wall and maximum width is less than 5 mm) (4). None of the AS patients had a history of sputum production for more than 3 months for 2 successive years, or any symptoms or clinical, radiological or electrocardiographic signs of a heart disease. All patients with LVF had dysponea [New York Heart Association (NYHA)] grade 2 or more (5), with left ventricular ejection fraction less than 45% as determined by X-ray contrast ventriculography, applying the standard formula of Sandler and Dodge (6). In all cases LVF was due to coronary artery disease. None of the LVF patients had any clinical or radiological evidence of pulmonary disease, and in particular none had a history of chronic sputum production or of occupational exposure to asbestos. Patients with ARPD had substantial asbestos exposure. Pleural disease was more than 2b (the length of pleural disease is more than one quarter of the lateral chest wall on a posteroanterior chest radiograph and the maximum width of pleural disease is more than 5 mm) (4). On HRCT 13 patients had both diffuse pleural thickening and pleural plaques. Two patients had calcified pleural plaques only and two patients had diffuse pleural thickening only. There was no HRCT evidence of interstitial fibrosis in any of the patients.

Methods

THE LUNG SOUND SYSTEM

The lung sound system consists of a Knowles type BL-1670 ceramic electron microphone which incorporates its own field effect transistor (FET) buffer stage. This microphone can be attached to the chest wall by a double-sided adhesive tape. The frequency response of this microphone is flat to within 3 dB between 200 Hz and 5 kHz. The audio signal is subjected to initial amplification via an amplifier with a

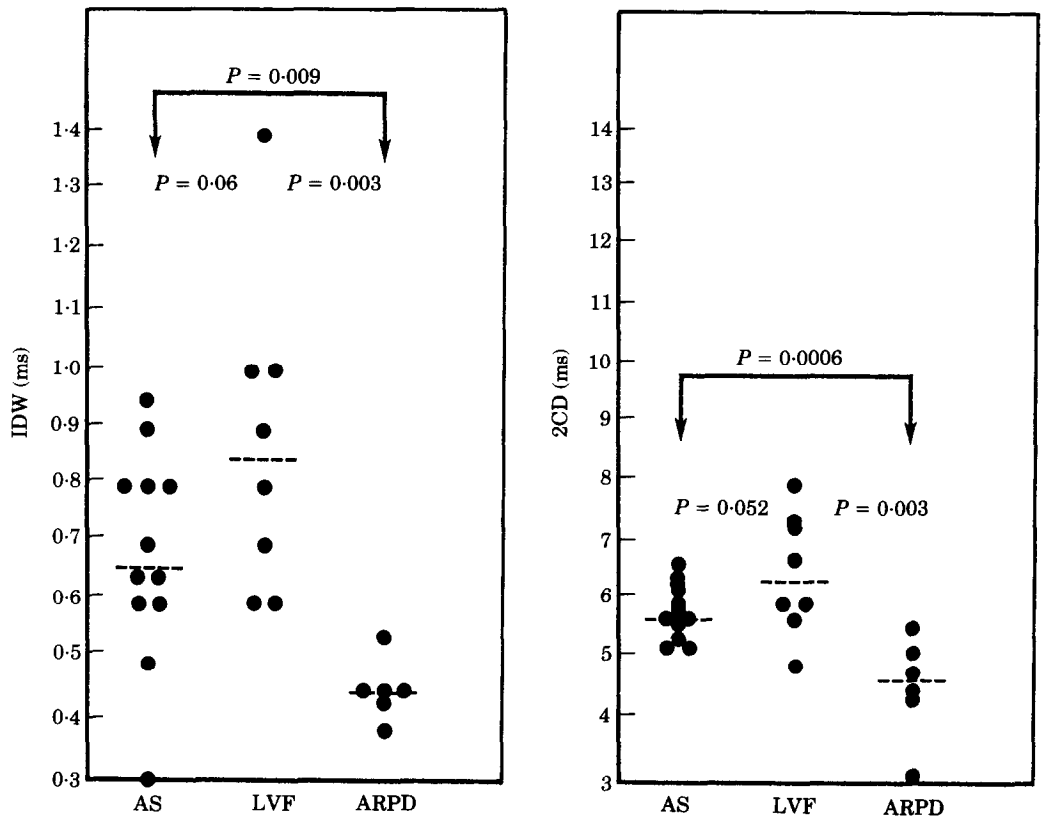

Fig. 2 IDW and 2CD for inspiratory crackles. Horizontal lines are the median values.

gain of 10. The sound signal is then filtered using an electronic filter EF5 (Fern Developments, formally Barr at Stroud Ltd). These filters can be set digitally to behave as a bandpass filter with a low and high pass roll of 4 dB per octave. The low pass filter 3dB-point was fixed at 2·5 kHz. The high pass filter 3dB-point was set at 100 Hz in order to cut out the low frequency sounds, particularly heart sounds. The sound signal can be seen on a real-time display and is sent to a multi-channel data acquisition board (type DT2821) installed in an IBM compatible PC, AT (Tandon). This board consists of a multi-channel A/D convertor. The sampling rate of each channel of the analogue to digital (A/D) converter is set at 10 kHz. A double buffered DMA approach was used to provide 33 s of gap-free samples.

Flow at the mouth is recorded using a pneumotachograph and low pressure transducer (type EMT 32c). The flow signal is displayed on the on-line screen. Two lines are shown on this screen and patients are asked to breathe so that the flow signal touches the upper line on peak inspiration and the lower line on peak expiration. The signal is further conditioned via an instrumentation amplifier, and its output is fed both to the real-time display, and also to the A/D converter. The flow signal and the sound signal can be superimposed for identification of the timing of events.

The software was developed using the scientific programming language 'ASYST'. The recording time is 33 s. For the purpose of TEW analysis a compressed display of 2·7 s can be produced by combining a number of subfiles. More detailed analysis can be performed on a window of a 100 ms.

THE SOUND RECORDING

Patients were rested for 15 min during which they were asked to cough repeatedly in order to clear any airway secretions. The microphone was placed on the posterior chest wall 4 cm below the inferior scapular angle. When hair was present the area where the microphone was placed was shaved. Recordings were made for all subjects while they were in the sitting position during quite deep respiration. Prior to recording patients were trained to breathe deeply and gently so that the flow line touched the two lines on the on-line display. The position and pattern of breathing were

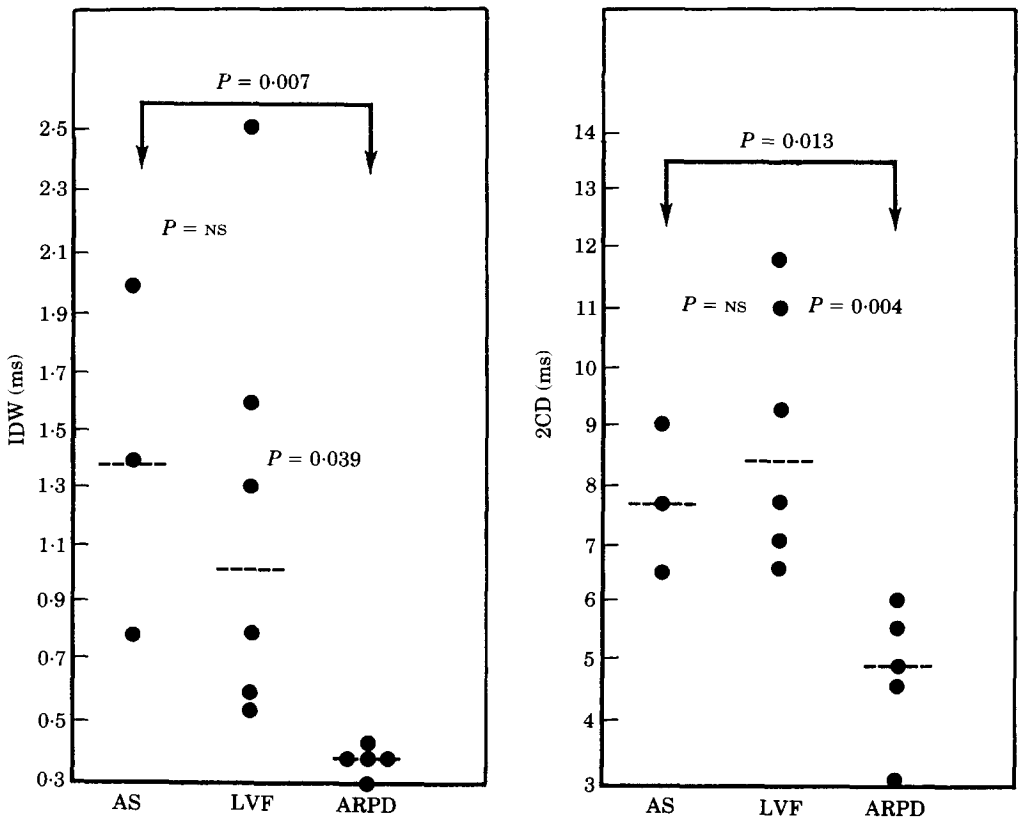

Fig. 3 IDW and 2CD for expiratory crackles. Horizontal lines are the median values.

chosen because they are similar to those used by clinicians when listening to lung sounds and because they were found to highlight complexes of adventitious lung sounds (crackles and wheeze) from background and vesicular sounds. It was found that other manoeuvres such as quiet tidal breathing or vigorous breathing from RV to TLC obscured abnormal lung sounds by reducing the amplitude of crackles in the former case and by disproportionately increasing the amplitude of vesicular lung sounds in the latter case.

DATA ANALYSIS

Crackles were considered to be present when two or more complexes were present during inspiration or expiration in the one cycle for at least three successive cycles.

A crackle was considered to be present when a sound complex met the following criteria (7): First, the amplitude of the largest peak was greater than double that of the background sound; second, the beginning of the event had a sharp negative or positive deflection and third, crossing of the base line by deflections was progressively wider.

A fine crackle was considered to be present when the initial deflection width (IDW) and the two cycle duration (2CD) were $<0{\cdot}92$ ms, $<6{\cdot}05$ ms respectively. A coarse crackle was considered to be present when IDW and 2CD were >1.25 ms and $>9{\cdot}23$ ms respectively (8,9).

Crackles which had intermediate figures were considered as medium crackles.

Three successive respiratory cycles from each recording were analysed. Variables chosen for analysis were: mean percentage of the total crackles that were inspiratory; time from the start of inspiration to the first inspiratory crackle as a percentage of the total inspiratory time; time from the start of inspiration to the last inspiratory crackle as a percentage of the total inspiratory time; time of the first expiratory crackle from the start of expiration as a percentage of the total expiratory time; time from the start of expiration to the last expiratory crackle as a percentage of the total expiratory time; the mean IDW for inspiratory and expiratory crackles in a respiratory cycle and the mean 2CD for inspiratory and expiratory crackles in a respiratory cycle.

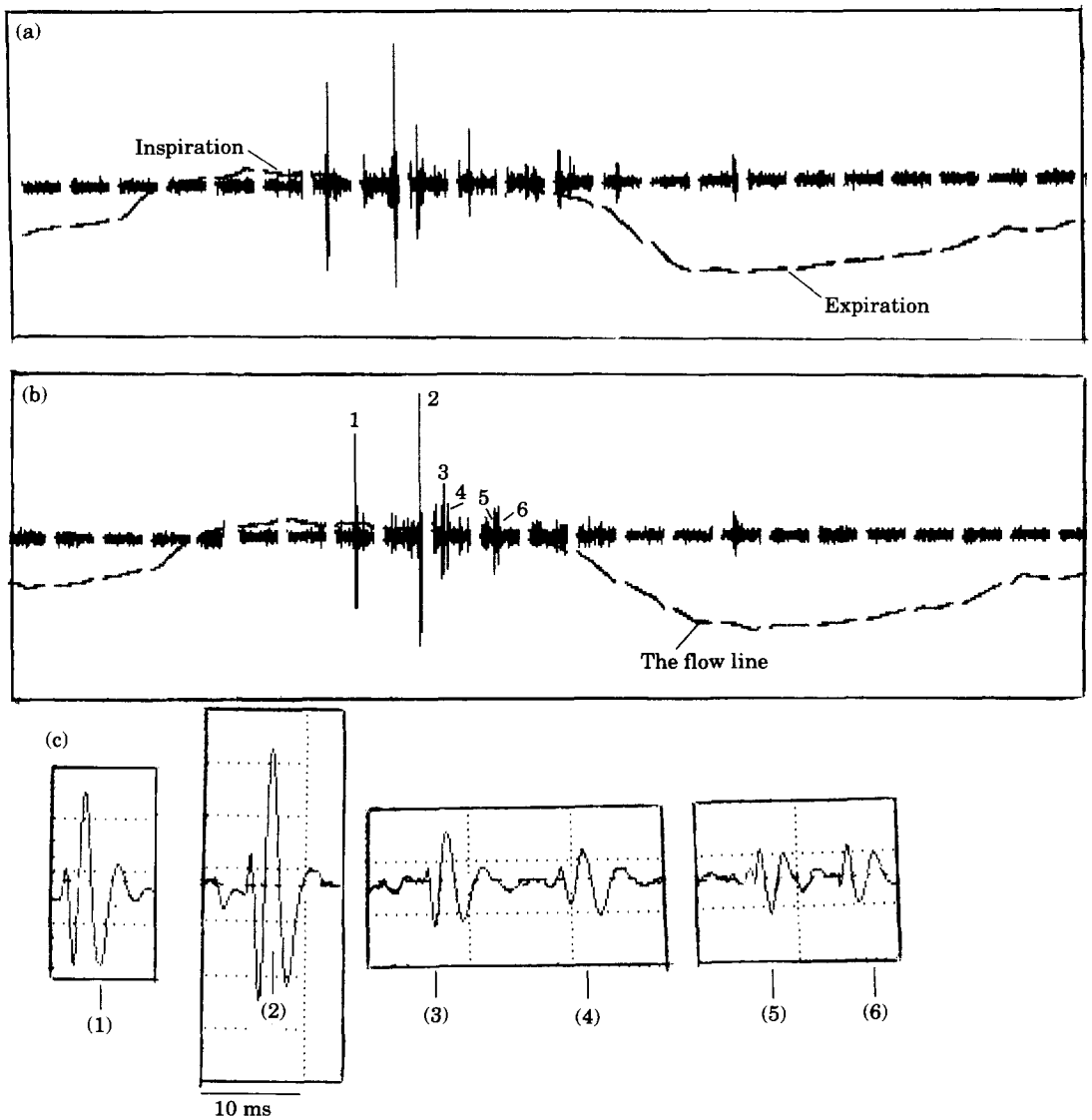

Fig. 4 Crackles in asbestosis [two respiratory cycles (a) and (b)]. Crackles are repetitive and the duration of any of them did not exceed 6·3 ms (2CD <6·3 ms) as seen in the detailed display (c).

The mean value of the three cycles for the first five variables for inspiration and expiration was taken for analysis.

STATISTICAL ANALYSIS

All statistical analyses were performed using Statgraph software (STSC). Measurements of IDW and 2CD were compared between the patient groups using a Mann–Whitney *U*-test.

Results

CRACKLE FEATURES

Table 1 shows the patients' demographic data and Table 2 shows the crackle features as means of the three successive cycles which were analysed. Crackle patterns in the three conditions were repeatable from one cycle to another and were reproducible over a short period (hours). Figs 1, 2 and 3 summarize crackle features in the three conditions.

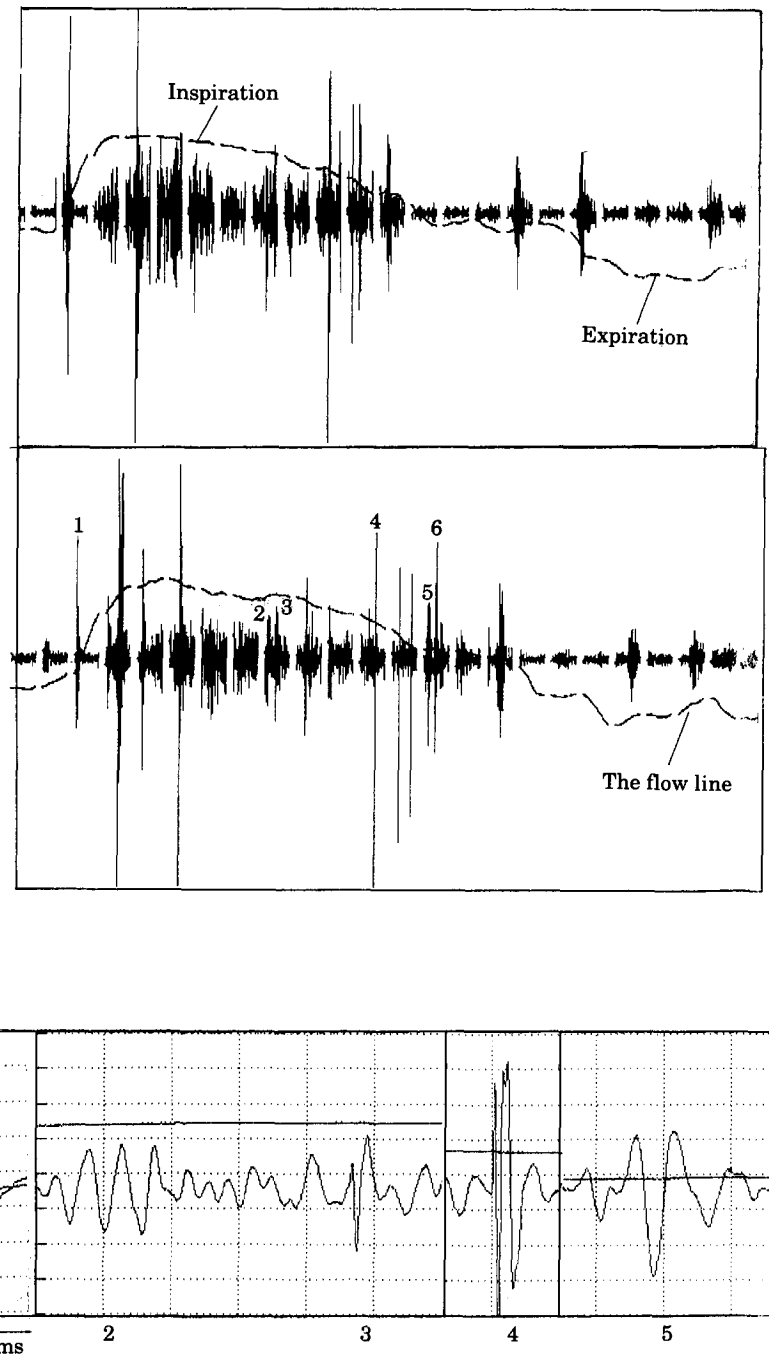

Fig. 5 Crackles in LVF, the first pattern. A crackle-free period is present in the middle of inspiration and the crackles are variable in duration. Selected crackles, shown in the detailed display, are either coarse (crackle 2 and 5) or fine (rest of crackles).

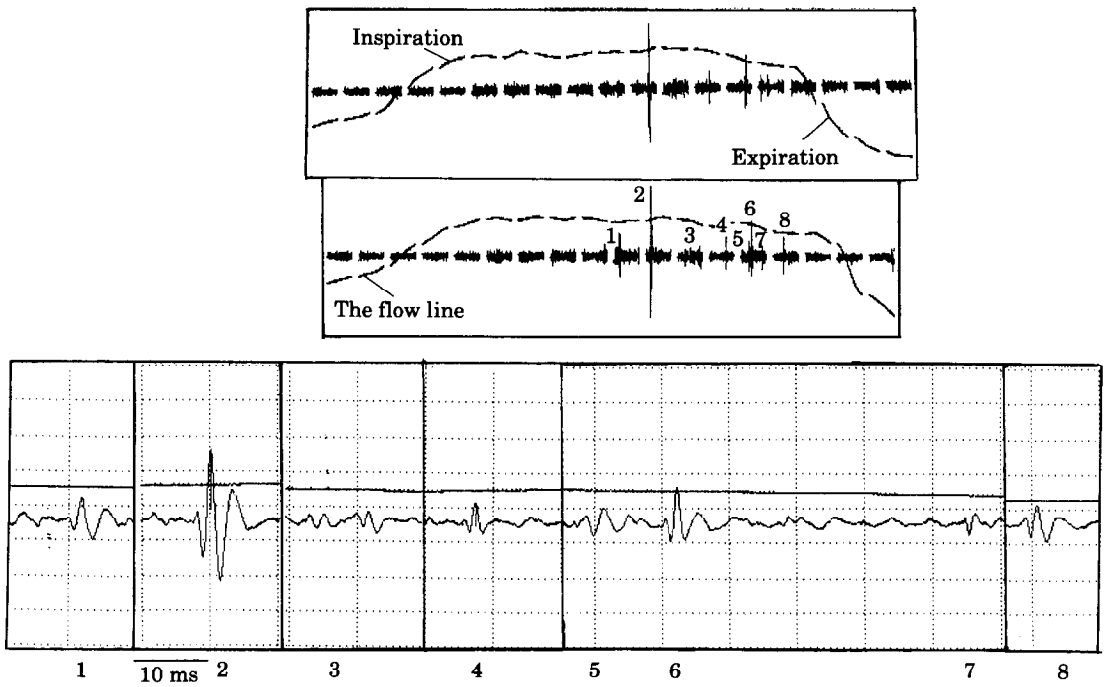

Fig. 6 Crackles in LVF, the second pattern. Recurrent end-inspiratory crackles. Unlike AS, crackles are fine (crackles 2, 4, 8), medium (crackles 1 and the second crackle in display 3) and coarse (the first crackle in display 3 and crackle 5).

ASBESTOSIS

Crackles were detected in all patients with AS. The distribution and the amplitude of the sequences of crackles were repetitive from cycle to cycle (Fig. 4). Ninety-two percent of all crackles were mid-to late-inspiratory. The expiratory crackles occurred in early to mid-expiration, Fig. 2. Inspiratory crackles were generally fine and expiratory crackles were coarse or medium (Table 2, and Figs 2 and 3).

LVF

In LVF crackles were detected by TEW in 9/11 patients. Sixty-three percent of crackles were inspiratory. Crackles were pan-inspiratory and early-to mid-expiratory, (Fig. 1). Crackles were of three patterns. In the first, crackles started early during inspiration followed by a crackle-free or minimum-crackle period then by another cluster of crackles lasting to the end-point of inspiration and continuing to almost mid-expiration (four patients), (Fig. 5). In the second, there were repetitive mid-to-late-inspiratory crackles similar to the pattern seen in AS except that there were coarse and medium as well as fine crackles (three patients), (Fig. 6). In the third, there were fine and medium expiratory crackles with few or no inspiratory crackles (two patients) (Fig. 7). In LVF IDW and 2CD were widely variable in inspiration and expiration (Table 2 and Figs 2 and 3).

ARPD

Crackles in ARPD were detected in 7/17 (41%) patients. Thirty-six percent of crackles were inspiratory. Crackles were fine mid-to late-inspiratory and fine early to mid-expiratory. Crackles generally took the configuration of crackles seen in asbestosis but another type of crackle was also present in all patients. It started with a sharp deflection (which looked like a pace-maker spike on the electrocardiograph) followed by an M-shape deflection then by the largest oscillation) (Figs 8 and 9). These crackles were repetitive and occurred during inspiration and/or expiration. They occurred in patients who had mainly diffuse pleural thickening under the area of sound recording and in one of the two patients who had calcified pleural plaques.

In ARPD both inspiratory and expiratory crackles were fine in nature; (mean values for IDW = 0·45, 2CD = 4·6 ms for inspiratory and IDW = 0·4, 2CD = 5·6 ms for expiratory crackles).

IDW and 2CD for inspiratory and expiratory crackles in AS tended to be shorter than those in LVF, but these values were shortest in ARPD.

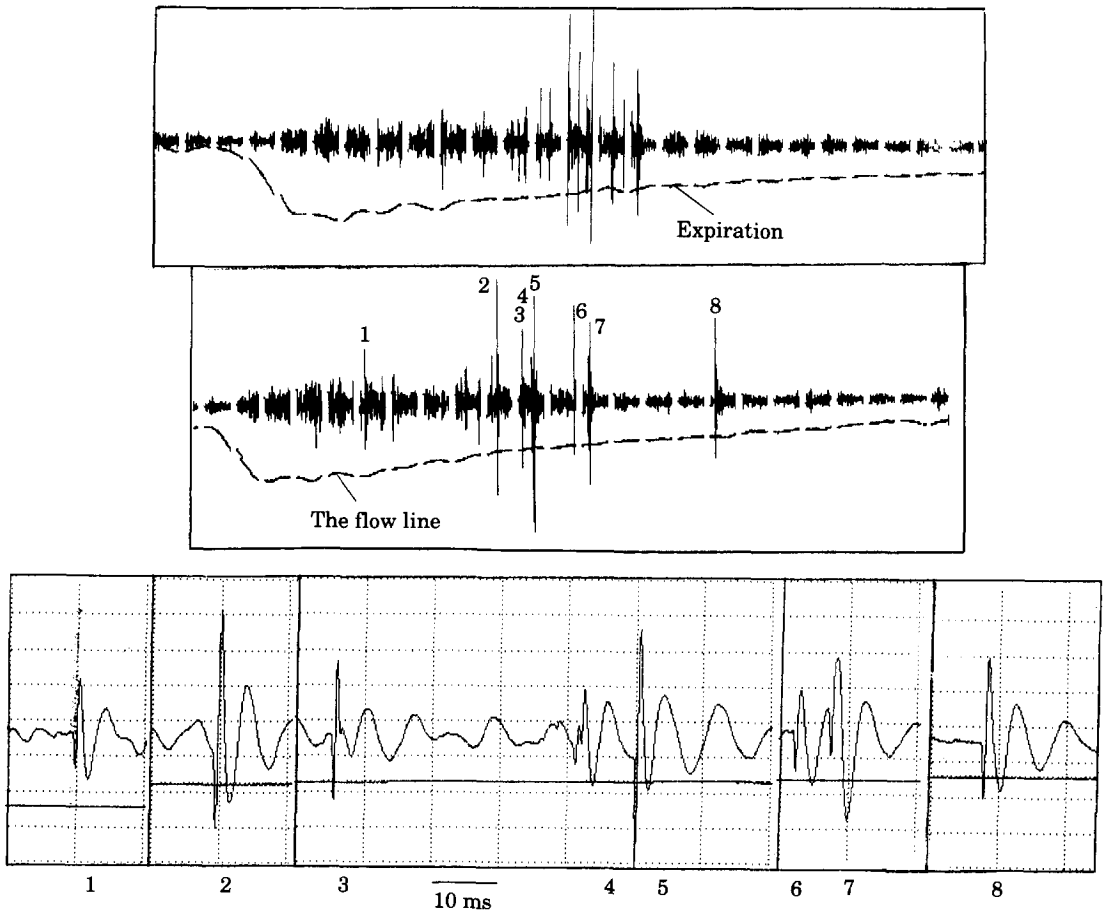

Fig. 7 Crackles in LVF, the third pattern. Recurrent expiratory crackles with no or minimal inspiratory crackles. Expiratory crackles are generally fine.

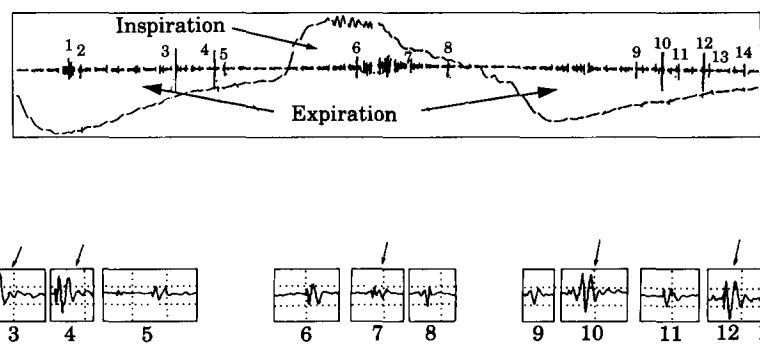

Fig. 8 Two respiratory cycles from recording of a patient with ARPD. Crackles are repetitive mid inspiratory and mid expiratory. Some crackles had different configuration from fine crackles (arrows).

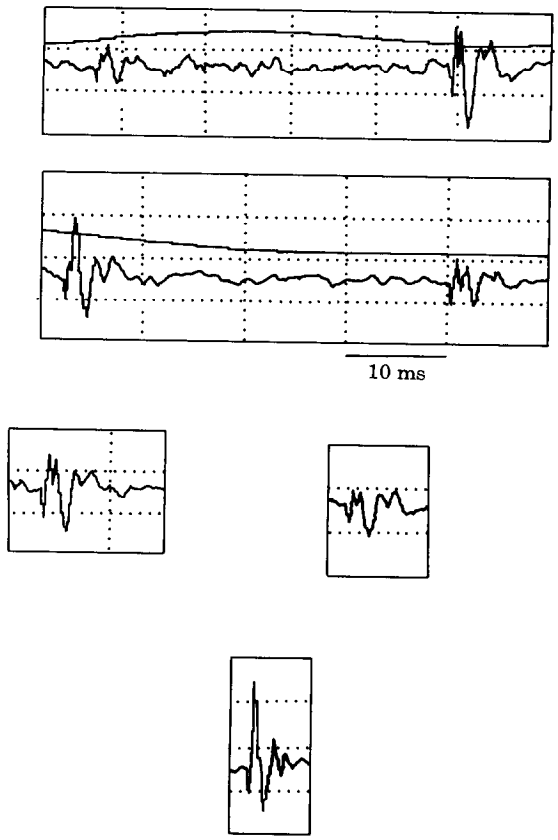

Fig. 9 Crackles from recordings of a patient ARPD (different from the patient in Fig. 8) Crackles preceded by a sharp deflection (similar to the pacemaker deflection seen on the electrocardiogram) followed by an M-shape oscillation followed by the oscillation of the largest amplitude.

Table 1 Demographic data and lung function tests expressed as predicted values for age, sex and height

	AS 12	LVF 11	ARPD 17
Mean age	59	59	54
Range	55–79	46–71	35–69
Male/female	12/0	11/0	15/2
Current smokers	4	1	4
Ex-smokers	8	9	10
Non-smokers	0	1	3
FEV_1			
mean	79	NA	79
SD	15		22
range	48–96		65–119
FVC			
mean	83	NA	79
SD	14		19
range	63–103		50–108
*T*LCO			
mean	73	NA	78
SD	22		17
range	52–118		61–99
*K*CO			
mean	88	NA	102
SD	25		13
range	53–139		83–123

NA, not available.

Discussion

In this study, different patterns of crackle clusters in patients with AS, LVF and ARPD have been demonstrated. Crackles in AS were repetitive in distribution and intensity. In a previous study, Nath and Capel (10,11) assumed that this repetitive pattern is more likely to be due to changes in solid structures rather than air bubbling through loose secretions. Expiratory crackles in AS accounted for less than 10% of all crackles and were fine to medium crackles. The mechanism of production of expiratory crackles is not clear.

In LVF a similar pattern to that seen in AS was noted in three of the nine patients but unlike asbestosis crackles in LVF tended to be medium and coarse as well as fine. Another pattern observed in LVF was that of pan-inspiratory crackles with a silent period in mid-inspiration associated with expiratory crackles. Inspiratory crackles in this pattern were fine and medium, whereas expiratory crackles were generally medium. A third pattern with only expiratory crackles was also noted in two patients. The second and third patterns are not seen in patients with asbestosis.

In a recent study crackles in LVF were mid inspiratory and medium to coarse in nature. Timing of expiratory crackles was not mentioned (12). Early inspiratory crackles could result from sudden opening by a downstream passage of gas of small airways lightly closed by an oedematous infiltration (3,12), whereas late inspiratory crackles might be caused by an abrupt forceful opening of more peripheral airways, each crackle representing the opening of a single airway (3,10,13,14). The inconsistent recurrence of expiratory crackles in this pattern suggests that the cause might be the passage of air through loose fluid in small airways which had not been cleared by cough prior to the study. Homma *et al.* (14) performed TEW, bronchoscopy and lung biopsies on animals while inducing progressive stages of pulmonary oedema. Lung crackles were fine in early stages when the biopsies showed interstitial oedema and when bronchoscopy revealed no fluid secretions in the airways. Lung crackles later became both coarse and fine when the biopsy showed interstitial and alveolar oedema and bronchoscopy revealed airways secretions. The cause of the third pattern of crackles obtained in LVF consisting of

Table 2 Crackles data. The figures are the mean of the figures obtained from the three respiratory cycles (Fig. 1)

	Inspiratory crackles					Expiratory crackles				
	No.	Start	End	IDW	2CD	No.	Start	End	IDW	2CD
Asbestosis ($n=12$)										
Mean	12	44	91	0·7	5·9	1	28	61	1·4	7·8
SD		13	10	0·2	0·4		13	16	0·6	1·2
LVF ($n=9$)										
Mean	8	36	86	0·6	6.6	4·7	26	70	1·24	8·9
SD		27	31	0·4	2·0		14	17	0·5	2·6
ARPD ($n=7$)										
Mean	4	58	90	0·45	4·6	5	29	72	0·4	4·8
SD		20	10	0·05	0·86		18	13	0·05	1·2

No., mean number of crackles; Start, time from the beginning of inspiration or expiration to the first crackle as a percentage of total inspiration for inspiratory crackles or expiration for expiratory crackles; End, time from beginning of inspiration or expiration to the last crackle taken as a percentage of the total inspiration or expiration; IDW, the mean initial deflection width for all inspiratory or expiratory crackles in one cycle; 2CD, the mean two-cycle duration for all inspiratory or expiratory crackles in one cycle.

expiratory crackles of short duration with few or no inspiratory crackles, is not clear.

In ARPD crackles were different in many respects from those which occurred in patients with asbestosis. Crackles in ARPD were fine both inspiratory and expiratory and many crackles had a characteristic configuration. The mechanisms of production of the crackles in ARPD and asbestosis are probably different. Crackles in asbestosis are thought to be produced by forceful opening of stiff peripheral airways and sudden equalization of pressure of air passing through small airways and alveoli. This mechanism is similar to the mechanism of crackle production by a paper or vinyl bag being inflated by air (3,13). Crackles in ARPD are believed to be produced by the friction resistance momentarily interrupting the sliding motion of the two layers of pleura. Forgacs (3) assumed that when the pleura is roughened by a fibrinous deposit or neoplastic cell, the lung acts on the chest wall like the bow of a string instrument. This explains the occurrence of crackles during mid inspiration and mid expiration when the maximum friction between the two layers occurs.

On TEW crackles were detected only in seven of 17 patients. The reason for this may be that crackles are very localized in nature and could not be detected at the lung bases where the recordings were performed.

We conclude that several differences exist in nature and pattern of distribution of lung crackles between AS, LVF and ARPD on TEW. This may reflect differences in the pathophysiology and mechanism giving rise to lung crackles in these conditions. TEW analysis of crackles can provide information of diagnostic value.

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